

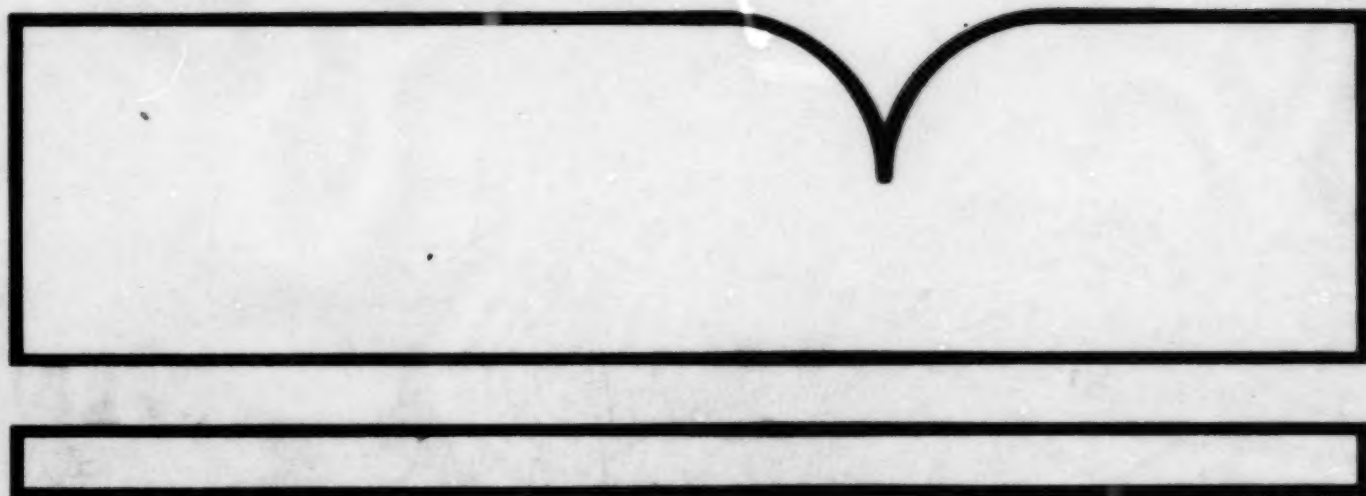
Asbestos in Drinking Water and Cancer
Incidence in the San Francisco Bay Area

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ASBESTOS IN DRINKING WATER AND CANCER INCIDENCE IN THE SAN FRANCISCO BAY AREA

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Age-adjusted, sex- and race-specific 1969-1971 cancer incidence ratios for the 722 census tracts of the San Francisco-Oakland Standard Metropolitan Statistical Area were compared with measured chrysotile asbestos counts in tract drinking waters. The water supplies serving the area have varying contact with naturally occurring serpentine. The *t* test for multiple regression coefficients and the *t* test for correlation coefficients showed significant ($p < 0.01$) relationships between chrysotile asbestos content of tract drinking water and white male lung, white female gall bladder and pancreas, and peritoneal cancers in both sexes. Of weaker significance ($0.01 \leq 0.05$) were female esophagus, pleura and kidney, as well as stomach cancers in both sexes. These associations appeared to be independent of income, education, asbestos occupation, marital status, country of origin and mobility.

asbestos; environmental pollutants; neoplasms; water pollutants; water supply

The occurrence of asbestos fibers in drinking water supplies became a subject of concern in 1973 when 1-74 million asbestos-like fibers per liter were reported in the Duluth, Minnesota drinking water supply (1, 2). The question of possible increased cancers in the general population resulting from long-term ingestion of asbestos fibers in drinking waters is of

importance because of the well-documented relationships between occupational asbestos exposures and increased respiratory and digestive cancers in exposed workers (3, 4).

In 1975, Mason et al. (5) reported a comparison of cancer mortality for Duluth with Hennepin County (which contains Minneapolis) and the entire state of Min-

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Abbreviations: BDL, below detectable limit; F/L, fibers per liter; ICD, International Classification of Diseases; LDL, lowest detectable limit; RCE, San Francisco Bay Area Resource for Cancer Epidemiology; SIR, Standard Incidence Ratio; SMSA, Standard Metropolitan Statistical Area; TNCS, Third National Cancer Survey.

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nesota. They used data collected in Mason and McKay's "US Cancer Mortality by County: 1950-1969" (6). They found that only cancer of the rectum was significantly higher ($p < 0.01$) for both sexes for Duluth. Levy et al. (7) reported in 1976 their comparisons of Duluth gastrointestinal cancer incidence rates for 1969-1971 with those of Minneapolis and St. Paul. The Minneapolis and St. Paul data were available from those compiled as part of the Third National Cancer Survey (TNCS). The investigators imitated the procedures of the TNCS to obtain their Duluth cancer incidence data, in order to avoid biasing the comparison. Generally, Minneapolis and St. Paul had higher gastrointestinal rates, but rates of cancer of the stomach, small intestine, and "peritoneum, retroperitoneum, and abdomen not otherwise specified" were higher in males in Duluth than in the other two cities. The incidence of cancers of the pancreas and "GI tract, not otherwise specified" were higher for both sexes in Duluth than in the comparison cities. Since the asbestos pollution of the Duluth water supply started in 1956 (2), and the latent period for certain asbestos-related tumors is as long as 40 years, not enough time had passed to draw final conclusions concerning the Duluth situation.

Wigle (8) in 1977 studied cancer mortality in relation to asbestos in municipal water supplies in 22 Quebec municipalities. He compared observed to expected 1965-1967 and 1970-1972 mortality for municipalities grouped by three classifications of exposure to asbestos in water supplies: known high, possible high and probable low. The expected numbers were calculated based on overall Quebec mortality rates. The municipalities with the known high exposure were located in areas where asbestos mining operations began in the last quarter of the 19th century. Rates of male lung cancer, male stomach cancer and female pancreatic cancer were significantly

higher in the known high exposure group than in the other two groups. Wigle attributed the male lung cancer finding to occupational asbestos exposure. The fact that male stomach cancer rates and pancreatic cancer rates for both males and females were also increased in Duluth (7) was suggestive that these two cancers may be related to asbestos fibers in drinking water supplies.

California has some of the richest deposits in the United States of the mineral serpentine, the parent rock form of chrysotile asbestos. In fact, serpentine is the official state rock of California (9). Several drinking water supplies in the San Francisco Bay area come from aquifers or are stored in reservoirs that are exposed to serpentine. The San Francisco-Oakland Standard Metropolitan Statistical Area (SMSA) was selected as the location for a study of the possible relationship between the presence of asbestos fibers in drinking water and the incidence of selected cancers in the populations served by such water supplies because there are several distinct water sources serving the SMSA, with differing amounts of asbestos in the drinking waters. The overall study design was that of a geographic correlation study. Census tracts with differing chrysotile asbestos counts in drinking water were compared for cancer incidence while controlling for potentially confounding factors. The San Francisco-Oakland SMSA contains five counties: Alameda, Contra Costa, Marin, San Francisco and San Mateo. The SMSA is divided into 722 census tracts and had a 1970 population of slightly over 3 million.

MATERIALS AND METHODS

Asbestos analysis

The average number of chrysotile asbestos fibers per liter of drinking water in each of the 722 census tracts in the SMSA had to be determined. It was first necessary to document the drinking water dis-

tribution systems of the five Bay area counties both present and historical. It was then necessary to select and take samples, analyze them for asbestos fiber count and assign a representative chrysotile fiber count to each census tract.

Meetings were held with the water suppliers to delineate the present and historical sources and distribution of each water supply system. This was necessary in order to obtain samples that would best typify the chrysotile asbestos exposure to the population via their drinking water over 40 years, the possible maximum latent period for asbestos-induced tumors. Generally, the area's drinking water sources consist of surface water originating in two distinct areas of the Sierra Nevada, Coast Range runoff, local groundwater and water from the Sacramento-San Joaquin Delta. Each census tract was coded as to which of the 74 water distribution companies served the tract and to the type of water that was the primary source of tract water. A sampling program was designed to determine the levels of chrysotile asbestos within each of the distribution systems. Analyses were made on raw and finished water at treatment plants, and from samples taken from various sites within a given distribution system, the latter to determine the variability of values throughout the distribution area. Most samples were taken from household taps. Stored emergency waters were also analyzed, some dating back to 1908. Although few of these water caches were available, they yielded some historical background data on the presence of asbestos in Bay area water.

In total, 353 water samples were analyzed for chrysotile asbestos. Analysis involved the filtration of each sample through a 0.45 μm pore size Millipore filter for examination and counting by transmission electron microscopy. The procedures and precision of the measurement techniques are described elsewhere (10). All analyses were done "blind"; each

sample was coded and then analyzed without the knowledge of source water. Several split samples were analyzed independently by another laboratory with almost identical results. Concentrations of chrysotile asbestos were recorded in fibers per liter (f/L) and ranged from below the detectable limit of the analysis (usually 10^4 f/L) to 1.8×10^6 f/L of finished drinking water.

Asbestos values were assigned to census tracts by calculating the arithmetic mean of finished and tap water samples from a given water distribution area and assigning that value to all the tracts in that service area. Where possible, each water district was subdivided by various sources, treatment processes and/or pressure zones.

When the sample result was below detectable limit (BDL), the lowest detectable limit (LDL) value of the analysis was used as the sample count in taking the mean. Using the LDL instead of zero was conservative in that it would tend to decrease differences in mean counts between tracts. Values were assigned to 705 census tracts out of the total 722 tracts in the study area. Most commonly, the remaining 17 tracts were served primarily by a series of private wells, and they involved private or small systems which could not be sampled because of changes over time that were too difficult to characterize.

In order to prevent possible bias, the assignment of asbestos values was conducted independently of the cancer incidence data. None of the personnel involved in calculating the asbestos values had knowledge of cancer incidence by census tract within the study area, and thus were not influenced in developing the methodology or any of the water distribution information used in the assignment process.

Tumor incidence

The dependent variable for this study

was the Standard Incidence Ratio (SIR). This is the ratio of observed to expected (O/E) number of cancer cases. In order to obtain the necessary SIR's, cancer incidence case data and 1970 census population data were used in an indirect age-adjustment procedure using standard rates, which in this case were calculated rates for the whole SMSA.

The cancer incidence case data for 1969-1971 was obtained from the San Francisco Bay Area Resource for Cancer Epidemiology (RCE), which is located in the State of California Department of Health Services. These data were collected as part of the TNCS, which collected medical and other data on the incidence of all newly diagnosed malignancies during the three-year period 1969-1971 for nine areas of the US. The areas included two entire states, Iowa and Colorado, and seven metropolitan areas, including the San Francisco-Oakland SMSA. The design and operations of the TNCS are detailed in a 1975 monograph (11).

Cutler et al. (12) in 1974 described the salient attributes of the data collected in this survey. The tumor site and type were coded according to the 1968 Manual of Tumor Nomenclature and Coding (13). Over 90 per cent of the cancers were microscopically confirmed (12). A noteworthy feature of the TNCS was the classification by morphologic type of tumor (sometimes called histologic type). This classification defined over 200 pathologically different cell types of malignancy (12). For each case, there were codes for age, sex, race, body site of the malignancy, histologic cell types, diagnosis date, hospital, method of diagnosis and census tract.

Basically, all major sites were analyzed except skin and bone. For purposes of analysis, some sites were combined into logical groupings. For instance, examples of these combination categories were all sites, the digestive tract, and colon plus rectum. In total, 35 individual sites and

groupings were prepared for analysis. See table 1 for a listing of these sites with both their RCE and 8th Revision International Classification of Disease (ICD) codes (14).

Population data for use with the three-year 1969-1971 cancer incidence data were taken from the 1970 Census of Population (15). The midpoint of that period (January 1, 1969-December 31, 1971) is June 30, 1970, which is very close to the date of the 1970 Census, April 1, 1970, allowing for direct use of the 1970 Census for obtaining population data for this study.

Age adjustment was performed by the indirect method. Observed numbers of cases for a site were compiled for five-year age groups in each tract for each sex-race group, and expected numbers were calculated by applying the SMSA age-, race-, sex-, site-specific rates as standard rates to the age, sex, race population distribution of each census tract. The race categories used were white, nonwhite and total population.

Other variables

Other variables were organized by census tract for inclusion in the analysis. These variables were picked because of their possible influence in confounding the relation between asbestos levels in drinking water and cancer incidence.

Socioeconomic status is well known to be related to cancer rates. For instance, stomach and lung cancers often appear to be associated with low and breast cancer with high socioeconomic status (16). The 1970 Census data on median family income and median school years completed for each census tract were used as indicators of socioeconomic status in the analysis. Since it is well known that married persons generally have lower disease rates than those who are not married (17), the numbers of each tract's population 14 years of age and over who were single, separated, widowed or di-

TABLE 1
Cancer sites analyzed in study of San Francisco Bay area asbestos in drinking water and cancer incidence, 1969-1971

Site no.	Site name	RCE site no.*	ICD 8th Revision site nos.†
1	All sites	010-940	
2	All digestive	500-590	150-159
3	Digestive tract	500-549	150-154
4	Esophagus	500-509	150
5	Stomach	510-519	151
6	Small intestine	520-529	152
7	Colon	530-539	153
8	Rectum	540-549	154
9	Digestive-related organs	550-589	155-158
10	Liver	550-551	155
11	Gall bladder	560-569	156
12	Pancreas	570-579	157
13	Peritoneum	580-589	158
14	Digestive organ NOS‡	590	159
15	All respiratory	619-639	161-163
16	Larynx	619	161
17	Trachea, bronchus, lung	620-621	162
18	Pleura	630	163.0
19	Mediastinum	631	163.1
20	Breast	700-709	174
21	Female reproductive	710-759	180-183
22	Cervix uteri	710	180
23	Corpus uteri	720	182.0
24	Ovary	750	183.0
25	Prostate	770	185
26	Urinary	800-810	188-189
27	Kidney	800-804	189
28	Bladder	810	188
29	Brain	930	191
30	Thyroid	940	193
31	Hodgkin's disease	010	201
32	Leukemia	040-049	204-208
33	Lung small cell carcinoma	621 RCE-hist 804	
34	Lung squamous	621 RCE-hist 807	
35	Lung adenoca NOS‡	621 RCE-hist 814	

* Source: Manual of Tumor Nomenclature and Coding (13). RCE = San Francisco Bay Area Resource for Cancer Epidemiology.

† Source: Eighth Revision International Classification of Diseases, 1968 (14).

‡ NOS = not otherwise specified.

forced in 1970 (all except married) were added together and divided by the tract total population 14 years of age and over to yield a variable called marital status.

A variable was prepared to estimate the numbers of workers in each tract who were employed in industries that might have exposure to asbestos. For each tract, the number of construction workers was

added to the number of electrical workers and textile workers and then divided by the total working population.

It is well known that site-specific cancer rates among many migrant population groups from other countries differ from those of the native US population (18-20). A list of countries for which migrant rates for specific sites exceed

those of the US native population was compiled from Haenszel (18), Kmet (19), and Lilienfeld et al. (20). Census data on native- and foreign-born persons of foreign stock or, in other words, first or second generation immigrants from each relevant country for a particular cancer site were divided by the total population of each census tract to form proportions between zero and one. When two or more countries were included for a site, the proportions of those countries were added forming a single index of amount of relevant foreign stock for each cancer site in question.

Data on residence stability and year moved into unit were also organized by census tract. They were both made in proportions to serve as indices of stability for the tracts. It was hoped that this variable would account for population mobility in the analysis.

RESULTS

Tract groupings by asbestos count

The first analysis step involved dividing the census tracts into groups according to their chrysotile asbestos count and then calculating the SIR for these groups. The object of the step was to determine if these observed to expected cancer ratios for each site showed a dose-response relationship with the assigned asbestos values. The results for white males and females are in tables 2 and 3.

The fiber count boundaries for the groupings were a compromise between having similar numbers of tracts in each grouping and having sufficient differences in the fiber count between groups. The Standard Incidence Ratios were tested for statistical significance using chi-square procedures as described by Fleiss (21). The SMSA race-, sex-specific crude rate for each cancer site was multiplied by the corresponding observed to expected ratios for each of the tract groupings. This resulted in indirect age-adjusted ratios. For each set of age-adjusted ratios, total, slope and linearity

chi-square tests were performed. The total chi-square equals the chi-square linearity plus the chi-square slope. The total chi-square tests for the statistical significance of the differences among the cancer rates. The slope chi-square tests for significant increasing or decreasing cancer rates from low to high asbestos groupings. The linearity chi-square tests whether or not increasing or decreasing Standard Incidence Ratios form a linear relationship. The chi-square total has three degrees of freedom; the chi-square linearity has two degrees of freedom; and the chi-square slope has one degree of freedom.

Table 4 lists the sites with significant chi-square slope results. It must be remembered that significant chi-square slope results can be obtained whether the gradient with asbestos being tested is positive or negative. Significant chi-square slope results were obtained from one site, corpus uteri, site No. 23 for white females, where the Standard Incidence Ratio gradient in relation was negative. The white and total populations appear to have almost identical results, due mostly to the fact that the Bay Area population is predominantly white. The small number of significant dose-response gradients for the nonwhite population is most likely due to the small population numbers in that group.

Effect of socioeconomic status

The effect of socioeconomic status was separated from that of asbestos by cross-classifying the census tracts into groups using these two variables. Thus, using three asbestos categories and three socioeconomic status indicator levels, the tracts were divided into nine groups. The observed/expected ratios were then calculated for each of the nine groups of tracts for each site for each sex-race group. This was done using two indicators of socioeconomic status, median school years completed and median family income.

TABLE 2

Observed/expected ratios of cancer incidence, by site, in white males, for census tract groupings, by chrysotile asbestos fiber counts in San Francisco Bay area, 1969-1971

Site no.	Site name	Chrysotile asbestos fiber/liter by tracts (total tracts = 705)			
		16,000- 290,000 (175 tracts)	300,000- 890,000 (246 tracts)	970,000- 15,000,000 (117 tracts)	16,000,000- 36,000,000 (167 tracts)
1	All sites	0.941	0.941	1.005	1.114
2	All digestive	0.911	0.923	1.003	1.159
3	Digestive tract	0.909	0.914	1.006	1.161
4	Esophagus	0.911	0.855	0.896	1.249
5	Stomach	0.845	0.928	1.066	1.175
6	Small intestine	0.864	1.142	0.647	1.421
7	Colon	0.994	0.900	1.023	1.126
8	Rectum	0.830	0.931	0.979	1.175
9	Digestive related organ	0.918	0.946	1.005	1.145
10	Liver	0.968	0.941	0.676	1.337
11	Gall bladder	0.864	0.978	1.138	1.049
12	Pancreas	0.954	0.928	1.051	1.106
13	Peritoneum	0.518	1.083	1.226	1.222
14	Digestive organ NOS*	0.888	1.062	0.731	1.253
15	All respiratory	0.919	0.938	0.965	1.165
16	Larynx	1.032	1.157	0.834	0.952
17	Trachea, bronchus, lung	0.909	0.935	0.966	1.173
18	Pleura	1.891	0.784	0.621	0.875
19	Mediastinum	0.547	0.861	1.717	1.126
20	Breast	1.163	0.921	1.134	0.804
21	Female reproductive				
22	Cervix uteri				
23	Corpus uteri				
24	Ovary				
25	Prostate	1.038	0.921	1.027	1.042
26	Urinary	0.887	1.020	1.057	1.036
27	Kidney	0.990	1.043	1.003	0.979
28	Bladder	0.845	1.010	1.078	1.057
29	Brain	0.811	0.994	1.148	1.067
30	Thyroid	0.872	1.063	0.865	1.220
31	Hodgkin's disease	1.041	0.909	1.078	0.967
32	Leukemia	1.100	0.901	0.975	1.067
33	Lung small cell carcinoma	1.052	0.978	0.970	1.009
34	Lung squamous	0.962	0.924	0.854	1.210
35	Lung adenocarcinoma NOS*	0.854	0.857	1.183	1.154

* NOS = not otherwise specified.

The data for only the white population were included since it appeared that the total population data were so similar to the data for the white population, and because of the relatively low number of nonwhite cases.

Tables 3-8 show the results of this cross-classification procedure for the body sites that showed significant ($p < 0.01$) chi-square slope results in table 4.

These results show that the apparent effect of asbestos exposure on the cancer ratios seen in the previous sections is maintained here irrespective of the effect of the socioeconomic status indicators. For example, for white male lung cancer (site No. 17), there appears to be a strong negative effect of socioeconomic status which is well known in the literature (16). Low income and education groups show more

TABLE 3

Observed/expected ratios of cancer incidence, by site, in white females, for census tract groupings by chrysotile asbestos fiber counts in San Francisco Bay area, 1969-1971

Site no.	Site name	Chrysotile asbestos fibers/liter by tracts (total tracts = 705)			
		16,000- 290,000 (175 tracts)	300,000- 890,000 (246 tracts)	970,000- 15,000,000 (117 tracts)	16,000,000- 96,000,000 (167 tracts)
1	All sites	0.986	0.972	1.023	1.036
2	All digestive	0.888	0.940	1.038	1.117
3	Digestive tract	0.922	0.937	1.028	1.100
4	Esophagus	0.638	0.840	1.015	1.431
5	Stomach	0.891	0.939	1.108	1.056
6	Small intestine	1.683	0.840	0.595	0.908
7	Colon	0.941	0.929	1.026	1.107
8	Rectum	0.902	0.975	1.012	1.062
9	Digestive-related organ	0.745	0.913	1.089	1.223
10	Liver	0.632	0.994	1.369	1.085
11	Gall bladder	0.892	0.648	1.016	1.374
12	Pancreas	0.761	0.015	1.039	1.144
13	Peritoneum	0.361	0.679	1.223	1.785
14	Digestive organ NOS*	0.940	1.359	0.948	0.730
15	All respiratory	0.956	0.986	1.009	1.058
16	Larynx	0.524	2.161	0.562	0.411
17	Trachea, bronchus, lung	0.962	0.985	1.003	1.009
18	Pleura		0.803		2.618
19	Mediastinum	1.226	0.287	1.929	1.046
20	Breast	1.002	0.966	1.095	0.986
21	Female reproductive	1.052	1.039	0.902	0.995
22	Cervix uteri	1.100	1.003	0.709	1.120
23	Corpus uteri	1.083	1.087	0.926	0.908
24	Ovary	0.973	0.991	0.990	1.062
25	Prostate				
26	Urinary	1.182	0.902	0.968	0.996
27	Kidney	1.164	0.854	0.898	1.099
28	Bladder	1.193	0.932	1.012	0.938
29	Brain	1.013	1.030	1.149	0.850
30	Thyroid	0.982	0.848	1.232	1.061
31	Hodgkin's disease	0.863	1.041	0.918	1.147
32	Leukemia	0.998	0.939	0.927	1.129
33	Lung small cell carcinoma	1.151	1.074	0.958	0.873
34	Lung squamous	0.945	0.871	1.069	1.143
35	Lung adenoca NOS*	0.949	1.020	1.080	0.970

* NOS = not otherwise specified.

lung cancer than do high income and education groups. However, in each individual socioeconomic indicator category there appears to be higher cancer ratios for higher asbestos. Other well-known socioeconomic gradients (16) that were substantiated in these results were sites 5 stomach (negative gradient), 22 cervix uteri (females, negative gradient), and 20 breast (females, positive gradient). It ap-

pears that the effect of asbestos is consistently observed at each level of socioeconomic status.

Multiple regression and correlation coefficients

Multiple regression analysis procedures were used to examine the relationship between census tract asbestos fiber values and cancer incidence while taking

TABLE 4

One-tailed, chi-square significance levels for slope by cancer site, race, sex and asbestos level, San Francisco Bay area, 1969-1971

Site no.	Site name	Significance levels					
		Whites		Nonwhites		Combined races	
		Males	Females	Males	Females	Males	Females
1	All sites	0.000001	0.05			0.000001	
2	All digestive	0.000001	0.00001			0.000001	0.00001
3	Digestive tract	0.000001	0.001			0.00001	0.001
4	Esophagus		0.01				0.001
5	Stomach	0.01				0.05	
6	Small intestine						
7	Colon	0.05	0.01				0.01
8	Rectum						
9	Digestive-related organs		0.0001			0.001	0.001
10	Liver						
11	Gall bladder		0.01				0.01
12	Pancreas						
13	Peritoneum		0.01				0.01
14	Digestive organ NOS*						
15	All respiratory	0.00001				0.0001	
16	Larynx						
17	Trachea, bronchus, lung	0.000001				0.00001	
18	Pleura						
19	Mediastinum						
20	Breast						
21	Female reproductive				0.05		
22	Cervix uteri						
23	Corpus uteri		0.01				0.01
24	Ovary						
25	Prostate						
26	Urinary						
27	Kidney						
28	Bladder						
29	Brain						
30	Thyroid						
31	Hodgkin's disease						
32	Leukemia						
33	Lung small cell carcinoma						
34	Lung squamous	0.01				0.05	
35	Lung adenocarcinoma NOS*					0.05	

* NOS = not otherwise specified.

into account many other variables. A log-linear model was used since the theoretical construction of the probability of developing cancer by a certain time yields a function of the log form. The dependent variable was the tract sex-, race-, site-specific Standard Incidence Ratio (the observed to expected ratio). For some of the body sites with low incidence (peritoneum, pleura, mediastinum, etc.) many

census tracts had zero observed cases. To avoid taking the antilog of zero in the regression, a small constant, 0.01, was added to each tract's observed to expected ratio. The independent variables used were median family income, median school years completed, marital status, asbestos industry workers, country of origin for certain sites, and tract asbestos value. The log-linear multiple regressions

TABLE 5

Observed/expected ratios of cancer incidence in white males by cancer tracts grouped by asbestos levels and education levels for chi-square slope significant ($p < 0.05$) sites, San Francisco Bay area, 1960-1971

Site no.	Site name and education level ¹	Asbestos level ²		
		Low	Medium	High
1	All sites			
	Low	0.994	0.968	1.149
	Medium	0.974	0.946	1.128
	High	0.998	0.930	0.990
2	All digestive			
	Low	1.008	0.934	1.220
	Medium	0.991	0.898	1.149
	High	0.933	0.925	1.039
3	Digestive tract			
	Low	0.978	0.961	1.219
	Medium	0.997	0.882	1.147
	High	0.817	0.896	1.041
5	Stomach			
	Low	1.036	1.088	1.366
	Medium	0.948	0.775	1.180
	High	0.655	0.857	0.939
15	All respiratory			
	Low	1.191	1.103	1.195
	Medium	0.976	0.966	1.194
	High	0.725	0.805	0.965
17	Trachea, bronchus, lung			
	Low	1.187	1.185	1.391
	Medium	0.976	0.877	1.190
	High	0.712	0.795	0.915
34	Lung squamous			
	Low	1.479	1.190	1.353
	Medium	0.960	0.817	1.133
	High	0.706	0.739	0.854

* Education levels, by median school years completed: Low, 5.2-12.3 years; Medium, 12.3-12.6 years; High, 12.7-17.0 years.

† Asbestos levels, by chrysotile fibers/liter: Low, 10,000-32,000; Medium, 330,000-4,100,000; High, 5,400,000-50,000,000.

‡ Tract distribution, by education and asbestos levels:

Education level	Asbestos level			Total tracts
	Low	Medium	High	
Low	87	103	73	263
Medium	84	98	78	260
High	69	93	74	240
Total tracts	190	294	225	709

were performed using the Statistical Package for the Social Sciences (21) packaged computer program. The regressions were run for white males and females. Table 9 lists the body sites with significant asbestos regression coefficients.

For both sexes, the only site that had a strong relationship between asbestos and cancer incidence ($p < 0.01$) was site 13 peritoneum. A strong relationship was statistically significant for white male site 17 lung cancer ($p < 0.001$) and for white female site 11 gall bladder ($p =$

TABLE 6

Observed/expected ratios of cancer incidence in white females by census tracts grouped by asbestos levels and education levels for chi-square slope significant ($p < 0.01$) sites, San Francisco Bay area, 1969-1971

Site no.	Site name and education level*	Asbestos level*		
		Low	Medium	High
2	All digestive			
	Low	0.885	0.972	1.167
	Medium	0.783	0.912	1.115
	High	0.946	0.953	1.059
3	Digestive tract			
	Low	0.908	0.991	1.132
	Medium	0.841	0.915	1.134
	High	0.948	0.908	1.034
4	Esophagus			
	Low	0.670	0.954	1.835
	Medium	0.182	0.666	1.148
	High	0.928	1.085	1.024
7	Colon			
	Low	0.988	0.975	1.056
	Medium	0.790	0.898	1.158
	High	0.988	0.932	1.060
9	Digestive-related organs			
	Low	0.780	0.836	1.355
	Medium	0.519	0.900	1.083
	High	0.863	1.111	1.141
11	Gall bladder			
	Low	0.953	0.808	1.599
	Medium	0.321	0.649	1.447
	High	1.169	0.478	1.021
13	Peritoneum			
	Low		0.253	1.373
	Medium	0.880	0.734	0.906
	High	0.255	1.581	2.192
23	Corpus uteri			
	Low	0.984	0.807	0.772
	Medium	0.925	1.043	0.818
	High	1.224	1.342	1.036

* See footnotes to table 5 for explanation of "Low," "Medium" and "High" levels, as well as tract distribution by education and asbestos levels.

0.001) and site 12 pancreas ($p = 0.005$). The asbestos variable was of marginal significance ($0.01 \leq p < 0.05$) for site 5 stomach for both sexes, and sites 4 esophagus, 18 pleura and 27 kidney for females.

The square of R , the multiple correlation coefficient, was low for these regression equations. It was always less than 0.10, usually from 0.005 to 0.05. This means that only a small proportion of the total variation in cancer incidence was

"explained" by the variables in the equations. Because of low multiple correlation, the regression data must be viewed with caution. It is likely that observations of census tracts with zero cases contributed to the low R values. The analysis was performed in spite of this, to remove the effects of covariables. Also, it should be pointed out that in the regression procedures as many as 210 regression equations were computed. Therefore, the overall Type I error for the study must be con-

TABLE 7

Observed/expected ratios of cancer incidence in white males by census tracts grouped by asbestos levels and income levels for chi-square slope significant ($p < 0.01$) sites, San Francisco Bay area, 1969-1971

Site no.	Site name and income level†‡	Asbestos level†‡		
		Low	Medium	High
1	All sites			
	Low	0.961	0.958	1.195
	Medium	0.986	0.976	1.067
	High	0.878	0.911	1.009
2	All digestive			
	Low	0.964	0.908	1.246
	Medium	0.906	0.931	1.082
	High	0.874	0.915	1.085
3	Digestive tract			
	Low	0.936	0.936	1.226
	Medium	0.917	0.924	1.082
	High	0.879	0.874	1.123
5	Stomach			
	Low	1.210	1.061	1.437
	Medium	0.636	0.813	1.211
	High	0.794	0.880	0.945
15	All respiratory			
	Low	1.034	1.060	1.340
	Medium	0.980	1.026	1.112
	High	0.794	0.792	0.852
17	Trachea, bronchus, lung			
	Low	1.023	1.067	1.342
	Medium	0.986	1.017	1.111
	High	0.771	0.799	0.861
34	Lung squamous			
	Low	1.421	1.101	1.475
	Medium	1.021	0.992	1.077
	High	0.704	0.765	0.691

* Income levels, by median family income: Low, \$2455-10,304; Medium, \$10,316-12,799; High, \$12,813-34,530.

† Asbestos levels, by chrysotile fibers/liter: Low, 16,000-320,000; Medium, 330,000-4,100,000; High, 5,400,000-36,000,000.

‡ Tract distribution, by income and asbestos levels:

Income level	Asbestos level			Total tracts
	Low	Med	High	
Low	57	94	78	229
Medium	57	94	78	229
High	65	100	71	236
Total tracts:	179	288	227	694

sidered in context. From a conservative standpoint, p values would have to be considerably smaller than say 0.05 or 0.01 in order to be considered significant. The consistency of the relationship for certain

sites across procedures lessens the concern for this issue, however.

Table 10 shows product-moment correlation coefficients between asbestos values and cancer rates by sites for asbestos

TABLE 8

Observed/expected ratios of cancer incidence in white females by census tracts grouped by asbestos levels and income levels for chi-square slope significant ($p < 0.01$) sites, San Francisco Bay area, 1969-1971

Site no.	Site name and income level*	Asbestos level†		
		Low	Medium	High
2	All digestive			
	Low	0.879	0.921	1.195
	Medium	0.825	0.989	1.035
	High	0.973	0.909	1.119
3	Digestive tract			
	Low	0.931	0.924	1.202
	Medium	0.861	0.993	1.020
	High	0.982	0.876	1.098
4	Esophagus			
	Low	0.855	0.977	2.178
	Medium	0.593	0.764	0.795
	High	0.628	0.976	1.105
7	Colon			
	Low	0.978	0.958	1.140
	Medium	0.849	0.949	1.030
	High	1.014	0.889	1.140
9	Digestive-related organs			
	Low	0.663	0.862	1.219
	Medium	0.670	0.940	1.091
	High	0.936	1.047	1.233
11	Gall bladder			
	Low	0.624	0.792	1.321
	Medium	0.872	0.610	1.460
	High	1.232	0.528	1.161
13	Peritoneum			
	Low		0.267	0.775
	Medium	0.937	0.719	1.546
	High		1.565	2.027
23	Corpus uteri			
	Low	1.003	0.708	0.812
	Medium	1.083	1.178	0.793
	High	1.127	1.247	1.059

* See footnotes to table 7 for explanation of "Low," "Medium" and "High" levels, as well as tract distribution by income and asbestos levels.

by sex for the white population. The problem of zero cases in census tracts for low incidence cancer body sites also entered the correlation coefficient analysis. Many census tract zero cancer rates for a low incidence site would tend to give lower correlations than would be obtained with higher incidence cancer sites. The t statistic for testing the null hypothesis that the sample correlations were drawn from a population with zero correlation was computed in each case. Significant coefficients are noted for sites with $p < 0.05$.

These significant sites include all of the significant sites found under regression procedures as well as some other sites. The other sites do not show up as significant in the regression since the regressions remove the effects of covariables.

Population mobility

It was necessary to consider population mobility in this study. The movement of the population into, out of, and within the Bay area could conceivably bias the data and result in spurious results, or obscure

TABLE 9
Cancer sites in white males and females with significant ($p < 0.05$) positive asbestos regression coefficients, San Francisco Bay area, 1969-1971

Site no.	Site name	p value	R squared
<i>White males</i>			
1	All sites	0.043	0.055
5	Stomach	0.044	0.033
13	Peritoneum	0.002	0.018
15	All respiratory	0.001	0.096
17	Trachea, bronchus, lung	<0.001	0.099
<i>White females</i>			
4	Esophagus	0.020	0.039
5	Stomach	0.024	0.024
9	Digestive-related organs	0.004	0.016
11	Gall bladder	0.001	0.019
12	Pancreas	0.005	0.018
13	Peritoneum	0.009	0.014
18	Pleura	0.010	0.012
23	Corpus uteri*	0.025	0.016
27	Kidney	0.021	0.013

* Negative association.

real differences in cancer incidence associated with exposure to asbestos in drinking water. Population mobility is always a factor in studies comparing different geographic areas, but it could be especially important in cancer studies due to the long latent period between initial exposure to the risk factor and onset of the disease.

It was decided to take out the tracts that were unstable with reference to residence stability to see how this would affect the results of the log-linear multiple regression analysis. If the results of this analysis that used only the most stable tracts remained similar to the analysis that used all the tracts, then this would in some sense remove residence instability, and, indirectly, population mobility as potential confounding factors.

Those tracts that had below 47 per cent of the population five years of age and over living in the same house in 1970 as in 1965 were removed, leaving 385 (of the original 705) so-called "high-stability" tracts for the analysis. Log-linear multiple regression was run identically to that

discussed in the Multiple Regression Section using only these 385 tracts.

The body sites with significant ($p < 0.05$) regression coefficients and correlation coefficients for the asbestos variable are shown in table 11. Again, note that fewer sites are indicated as being significant using regression procedures since the regressions remove the effects of covariables. For the high stability tracts, site 13 peritoneum for both sexes, and site 11 gall bladder for females were statistically significant. Site 27 kidney for females and site 4 esophagus for males were of less certain significance.

DISCUSSION

A major assumption of this study was that the asbestos measurements taken in 1977-1978 were reflective of similar asbestos levels over the past 30 or 40 years. The major California drought, which started in the winter of 1975 and continued until early 1978, has to be considered in evaluating the results of this study. It is uncertain as to the effect of low rainfall on the relative order of asbestos

TABLE 10

Product-moment correlation coefficients of cancer incidence rates by asbestos values by site for white males and females, San Francisco Bay area, 1969-1971

Site no.	Site name	White males		White females	
		Correlation coefficient	p value*	Correlation coefficient	p value*
1	All sites	0.115	<0.01	0.091	<0.02
2	All digestive	0.107	<0.01	0.091	<0.05
3	Digestive tract	0.083	<0.05	0.049	
4	Esophagus	0.086	<0.05	0.116	<0.01
5	Stomach	0.059		0.077	<0.05
6	Small intestine	0.039		0.019	
7	Colon	0.065		0.010	
8	Rectum	-0.008		-0.007	
9	Digestive-related organs	0.019		0.117	<0.01
10	Liver	0.020		0.026	
11	Gall bladder	0.006		0.128	<0.01
12	Pancreas	0.004		0.105	<0.01
13	Peritoneum	0.108	<0.01	0.106	<0.01
14	Digestive organ NOS†	0.029		0.001	
15	All respiratory	0.097	<0.02	0.032	
16	Larynx	-0.019		0.020	
17	Trachea, bronchus, lung	0.108	<0.01	0.033	
18	Pleura	-0.014		0.102	<0.01
19	Mediastinum	0.027		-0.001	
20	Breast	0.004		0.061	
21	Female reproductive			-0.067	
22	Cervix uteri			-0.024	
23	Corpus uteri			-0.067	
24	Ovary			0.055	
25	Prostate	0.028			
26	Urinary	-0.006		0.028	
27	Kidney	-0.053		0.079	<0.05
28	Bladder	-0.011		0.012	
29	Brain	-0.011		0.008	
30	Thyroid	0.039		0.077	<0.05
31	Hodgkin's disease	0.038		0.017	
32	Leukemia	0.019		0.074	
33	Lung small cell carcinoma	-0.002		-0.015	
34	Lung squamous	0.023		0.060	
35	Lung adenoca NOS†	0.069		0.035	

* p value based on t test for $H: p = 0$.

† NOS = not otherwise specified.

N = 705 census tracts.

counts among the Bay area water districts. In areas where asbestos fibers are picked up from erosion of serpentine watershed, reduced runoff may have resulted in lower asbestos counts. In other areas it is conceivable that counts generally could have been higher during the drought, due to lack of dilution. However, comparisons with previous years' data

and samples gathered after the drought indicate little effect in asbestos values. Another year of sampling, with particular emphasis on possible seasonal variations in asbestos counts, would add certainty to these data. The history of the water supply and distribution systems was carefully documented. The major water supplies have not changed to a great ex-

TABLE 11

High stability census tracts: Cancer sites in white males and females with statistically significant ($p < 0.05$) asbestos regression coefficients and correlation coefficients, San Francisco Bay area, 1969-1971

White males				White females			
Site no.	Site name	p value	R squared	Site no.	Site name	p value	R squared
<i>Regression coefficients</i>							
4	Esophagus	0.019	0.045	11	Gall bladder	0.003	0.028
13	Peritoneum	0.003	0.040	13	Peritoneum	0.003	0.026
				23	Corpus uteri*	0.003	0.045
				27	Kidney	0.027	0.028
<i>Correlation coefficients</i>							
1	All sites	<0.05		11	Gall bladder	<0.01	
2	All digestive	<0.01		13	Peritoneum	<0.01	
4	Esophagus	<0.01		27	Kidney	<0.05	
13	Peritoneum	<0.01					
17	Trachea, bronchus, lung	<0.05					

* Negative association.

tent over the last 40 years. In most cases, it appears that the asbestos is present as a result of water passing through reservoirs or through other areas that contain serpentine rock. This basic circumstance has not changed over time.

Samples were taken before and after passage of water through lengths of asbestos-cement pipe. In most of the districts sampled, there was not a substantial increase in fiber counts after passage through asbestos-cement pipe. The amount of asbestos-cement pipe in the various distribution systems was not included as a variable in the analysis because of the difficulty of determining its location, age and dimensions. This study compared areas with differing amounts of asbestos fibers in drinking water from any source, be it natural, as from serpentine formations, or from asbestos-cement pipe.

Asbestos air pollution was a potentially confounding variable to consider. Ambient air samples were taken in each of the five study counties. All were below two fibers per liter of air. An air sample was also taken in the laboratory room where the water filtering and grid preparation were performed. This measure-

ment was below the detectable limit of one fiber per liter of air.

A characteristic of the study findings is the differential results by sex. The major differences by sex were positive relationships with chrysotile asbestos fiber counts in water for lung cancer in white males but not females, and cancers of the gall bladder, pancreas, pleura and kidney in white females, but not males. Stomach and peritoneal cancers were positively related to chrysotile asbestos in both white males and females. Overall, in evaluating the sex differences in the results of studies of potential carcinogenic agents, it must be remembered that these types of differences have been well documented previously. For instance, it is known that chloroform causes kidney cancer in male, but not female mice (23).

One could speculate that a differential exposure to chrysotile asbestos by sex could possibly explain these different results for males and females. For instance, females could be more consistently exposed to asbestos fibers in drinking water at their place of residence perhaps because they are at home more than males. This study was based on place of residence and did not account for daily mobility fac-

TABLE 12

Summary of statistically significant ($p < 0.5$) associations comparing asbestos and cancer site in white males and females, San Francisco Bay area, 1969-1971

Site no.	Site name	White male statistical test* p value			White female statistical test* p value		
		1	2	3	1	2	3
1	All sites	<0.01	<0.000001	0.043	<0.02	<0.05	
2	All digestive	<0.01	<0.000001		<0.05	<0.00001	
3	Digestive tract	<0.05	<0.000001			<0.001	
4	Esophagus	<0.05			<0.01	<0.01	0.02
5	Stomach		<0.01	0.044	<0.05		0.024
7	Colon		<0.05			<0.01	
8	Rectum						
9	Digestive-related organs				<0.01	<0.0001	0.004
11	Gall bladder				<0.01	<0.01	0.001
12	Pancreas				<0.01		0.005
13	Peritoneum	<0.01		0.002	<0.01	<0.01	0.009
15	All respiratory	<0.02	<0.00001	0.001			
17	Trachea, bronchus, lungs	<0.01	<0.000001	0.001			
18	Pleura				<0.01		0.01
24	Ovary						
27	Kidney				<0.05		0.021
30	Thyroid				<0.05		
32	Leukemia						
34	Lung squamous		<0.01				
35	Lung adenoca NOS†		<0.05				
23	Corpus uteri‡					<0.01	0.025

* Statistical test code: 1 = t test for correlation coefficient; 2 = chi-square slope; 3 = t test for regression coefficient.

† NOS = not otherwise specified.

‡ Negative association.

tors, such as place of work. Another possible hypothesis would be based on differential susceptibility of the sexes to asbestos in water with respect to carcinogenesis. Thus, increased male, but not female, lung cancer resulting from asbestos exposure via drinking water could be a result of more male exposure to cigarettes and occupational carcinogens, which could leave males more susceptible to the carcinogenic potential of ingested asbestos fibers which migrate to the lungs via the blood or through some other route.

A statistical association between asbestos fiber content in drinking water and the incidence of certain cancers in the San Francisco Bay area has been shown. A summary of the results of the chi-square test for positive slope, the t test for multiple regression coefficients, and the t test

for correlation coefficients is presented in table 12. All three tests for the white population indicated several cancer sites as being associated with chrysotile asbestos fibers in drinking water. Male lung and female gall bladder and peritoneum were consistently significant sites throughout the study. Male all-sites and female esophagus were associated with a lower level of significance ($p < 0.05$). Although not consistent for all three statistical tests, male peritoneum was significant to the $p < 0.01$ level, as well as female pancreas and stomach in both sexes in the log-linear regression analysis. Female pleura and kidney were significant at the $p < 0.05$ level. The fact that the significant results are not restricted to one body site might be explained by experimental results which

indicate that asbestos fibers are transported throughout the body (24-26).

It is interesting to compare these findings with the results of other studies. There appears to be a certain consistency of findings with results by Levy et al. (7) which compared 1969-1971 cancer incidence for Duluth with Minneapolis and St. Paul, and Wigle's study (8) of Quebec mortality rates. Male stomach and female pancreatic cancers were significant ($p < 0.05$) for both of these studies. When included as analysis sites in either study, male lung and peritoneal cancers were also suggestive.

The study design was that of an indirect epidemiologic approach in which the observational unit was the census tract. Studies such as this can only suggest associations and data on potentially important variables such as cigarette smoking, alcohol consumption and other dietary factors were unavailable.

An important concomitant variable, socioeconomic status, was available. The fact that the effect of asbestos is seen in all levels of socioeconomic status, helps lessen concern for confounding effects of occupation. It is reasonable to assume that most construction or shipyard workers who are exposed to asbestos are in the lower or middle socioeconomic categories. Since the Standard Incidence Ratios showed a positive dose-response gradient for asbestos in water in all socioeconomic groups, it is not likely that the observed associations can be entirely accounted for by occupational factors. Likewise, analyses of the high-stability tracts indicate that population mobility did not seriously bias the results.

Additional years of incidence data will be added to the data of this study for reanalysis. For some of the sites in the 1969-1971 analysis, such as peritoneum, the numbers of cases were insufficient for definitive study. Currently, the Bay area cancer incidence for 1972-1974 is being combined with the 1969-1971 data for a

reanalysis using the total 6-year 1969-1974 period. It is hoped to be able to analyze the data in the future using other water parameters such as chloroform, other organics and pesticide content.

Most importantly, it is recommended that a case-control study be conducted to further clarify the relationship between asbestos fibers in drinking water and cancer of certain sites. This type of study would compare individual cancer cases with controls for exposure to asbestos in drinking water. Additional information could be obtained by questionnaire on the residences, occupations and smoking and drinking habits of these individuals. A case-control study would lead to a more definitive answer as to the effects on cancer incidence of asbestos fibers in drinking water.

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